6/3,K,AB/4 (Item 4 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

09664878 98089587 PMID: 9428118

Donor left ventricular **hypertrophy** increases risk for early graft failure.

Aziz S; Soine L A; Lewis S L; Kruse A P; Levy W C; Wehe K M; Fishbien D P ; Allen M D

Department of Surgery, University of Colorado Health Sciences Center, Denver 80262, USA.

Transplant international : official journal of the European Society for Organ Transplantation (GERMANY) 1997, 10 (6) p446-50, ISSN 0934-0874 Journal Code: 8908516

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

6/25

A **review** of factors contributing to early mortality after **cardiac transplantation** revealed that up to 25% of deaths were due to primary graft dysfunction unrelated to rejection or infection. In light of this finding, evaluation of a donor heart with regard to its suitability for transplantation takes on added importance. In an effort to screen the suitability of donor hearts in the region covered by the Northwest Organ Procurement Agency (USA), all donors are evaluated by two-dimensional transthoracic echocardiography as part of the initial evaluation. A total of 110 donor echocardiograms were reviewed and an attempt was made to correlate the 30-day outcome with the parameters measured. An unexpected finding was that the presence of left ventricular **hypertrophy** in the donor heart was associated with an increase in the incidence of donor heart dysfunction compared with donors with normal echocardiographic profiles (33% vs 3%, P = 0.007).

Donor left ventricular **hypertrophy** increases risk for early graft

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 ds

Set     Items   Description
S1      58990   (CARDIAC OR HEART) (5N) TRANSPLANT?
S2     102995   HYPERTROPHY
S3       1009   S1 AND S2
? s review
      S4  642647  REVIEW
? s s3 and s4
           1009  S3
         642647  S4
      S5      32  S3 AND S4
? rd
>>>Duplicate detection is not supported for File 340.

>>>Records from unsupported files will be retained in the RD set.
...completed examining records
      S6      30  RD (unique items)
? t s6/3,k,ab/1-10

 6/3,K,AB/1     (Item 1 from file: 155)
DIALOG(R)File 155:MEDLINE(R)
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3/3,K,AB/3 (Item 3 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

13619398 21644600 PMID: 11784525

[Importance of tumor necrosis factor-alpha in the pathogenesis of heart failure]

Importancia del factor de necrosis tumoral alfa en la patogenia de la insuficiencia cardiaca.

Heberto Herrera Garza Eduardo; Herrera Garza Jose Luis; Rodriguez Gonzalez Humberto; Trevino Trevino Alfonso; Ibarra Flores Marcos; Torre Amione Guillermo

Departamento de Cardiologia del Centro de Medico del Noreste #34 del IMSS, Monterrey NL. Mexico.

Revista espanola de cardiologia (Spain) Jan 2002, 55 (1) p61-6, ISSN 0300-8932 Journal Code: 0404277

Document type: Journal Article; Review; Review, Tutorial ; English Abstract

Languages: SPANISH

Main Citation Owner: NLM

Record type: Completed

6/25

Clinical and experimental evidence demonstrating the effects of tumor necrosis factor-alpha (TNF-alpha) in patients with heart failure continues to accumulate. It is well established that high concentrations of TNF-alpha appear in the circulation of patients with heart failure and that these levels have a directly proportional correlation with the patient's functional class. TNF-alpha levels also show a linear relation with prognosis. These circulating levels are responsible for the decreased expression of myocardial TNF-alpha receptors observed in heart failure. As a result of extrapolation of findings from experimental animals, we assume that TNF-alpha is deleterious to myocardial function in humans because it induces a negative inotropic state in patients who have not undergone **heart transplant** . Supporting this assumption is the fact that the resolution or improvement of pressure overload (obstructive hypertrophic myocardiopathy, by ethanol ablation) and volume overload (terminal dilated myocardiopathy, by ventricular assistance) states is accompanied by a decrease in myocardial TNF-alpha expression.The use of specific antagonists of circulating TNF-alpha in patients with symptomatic heart failure has been demonstrated to be safe and possibly effective. At present, multicenter studies are under way to assess the efficacy of this antagonism in a larger number of patients. If the results of these studies are favorable, we will have new therapeutic elements for managing patients with advanced hear failure.The **transplanted heart** behaves differently from the native heart. From the early stages of HTx, myocardial TNF-alpha expression is greatly increased (much more than in patients with heart failure) and not associated with contractile dysfunction, in contrast with what occurs in the native **heart**. However, we know that the **transplanted heart** soon develops ventricular **hypertrophy**, fibrosis, diastolic dysfunction, and late graft failure, even in the presence of normal epicardial coronary arteries. Clinical evidence suggests that TNF-alpha may be involved in these processes.

... in humans because it induces a negative inotropic state in patients who have not undergone **heart transplant** . Supporting this assumption is the fact that the resolution or improvement of pressure overload (obstructive...

... favorable, we will have new therapeutic elements for managing patients with advanced hear failure.The **transplanted heart** behaves differently from the native heart. From the early stages of HTx, myocardial TNF-alpha...

... failure) and not associated with contractile dysfunction, in contrast with what occurs in the native **heart**. However, we know that the **transplanted heart** soon develops ventricular **hypertrophy**,

fibrosis, diastolic dysfunction, and late graft failure, even in the presence of normal epicardial coronary...
; Heart Failure, Congestive--drug therapy--DT; **Heart** Failure, Congestive--surgery--SU; **Heart Transplantation**; Myocardium --metabolism--ME; Receptors, Tumor Necrosis Factor--physiology--PH; Tumor Necrosis Factor--antagonists and inhibitors...

3/3,K,AB/4 (Item 4 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

13613608 22163453 PMID: 12173855
Echocardiographic evidence of right ventricular remodeling after transplantation.
Eltzschig Holger K; Mihaljevic Tomislav; Byrne John G; Ehlers Raila; Smith Brian; Shernan Stanton K
Department of Anesthesiology, Perioperative and Pain Medicine, Brigham and Women's Hospital, Harvard Medical School, Boston, Massachusetts 02115, USA.
Annals of thoracic surgery (United States) Aug 2002, 74 (2) p584-6, ISSN 0003-4975 Journal Code: 15030100R
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Right ventricular (RV) failure is a significant source of mortality after **cardiac transplantation**. The use of RV assist devices (RVAD) as a bridge to recovery has been reported. However, early changes of RV structure and anatomy after RVAD implantation have yet to be described. We report a case of RV failure after transplantation requiring RVAD implantation. After 3 weeks of

```
Set      Items   Description
S1      108544   HYPERTROPHY
S2       61641   (CARDIAC OR HEART) (5N) TRANSPLANT?
S3        1069   S1 AND S2
S4     4068562   DEVELOP?
S5         231   S3 AND S4
S6         165   S5 AND PY<=1998
S7         117   RD (unique items)
? s develop? (5n)hypertrophy
         4068562  DEVELOP?
          108544  HYPERTROPHY
      S8    5782  DEVELOP? (5N)HYPERTROPHY
? s s2 and s8
           61641  S2
            5782  S8
      S9      43  S2 AND S8
? rd
>>>Duplicate detection is not supported for File 340.

>>>Records from unsupported files will be retained in the RD set.
...completed examining records
     S10      26  RD (unique items)
? t s10/3,k,ab/1-26

 10/3,K,AB/1     (Item 1 from file: 155)
DIALOG(R)File 155:MEDLINE(R)
(c) format only 2003 The Dialog Corp. All rts. reserv.

11521627   98412546   PMID: 9741515
  Quantitative investigation of cardiomyocyte hypertrophy and myocardial
fibrosis over 6 years after cardiac transplantation.
  Armstrong A T; Binkley P F; Baker P B; Myerowitz P D; Leier C V
  Division of Cardiology, The Ohio State University College of Medicine,
Columbus, USA.
  Journal of the American College of Cardiology (UNITED STATES)  Sep 1998,
 32 (3) p704-10, ISSN 0735-1097  Journal Code: 8301365
  Document type: Journal Article
  Languages: ENGLISH
  Main Citation Owner: NLM
  Record type: Completed
  OBJECTIVES: This study was performed to determine the degree and time
course over 6 years of cardiomyocyte hypertrophy and myocardial fibrosis of
the cardiac allograft in transplanted patients. BACKGROUND:
Diastolic dysfunction and to a certain extent systolic dysfunction are
common cardiac findings after heart transplantation. The
development of posttransplant cardiomyocyte hypertrophy and
myocardial fibrosis likely contributes to these derangements. METHODS:
Cardiomyocyte diameter and percent fibrosis were determined in serial
endomyocardial biopsy specimens obtained from 1 month up to 6 years
following heart transplantation in 50 patients. Endomyocardial
biopsy specimens from 40 patients with primary dilated cardiomyopathy and
11 normal subjects were similarly analyzed for control data. Analyses were
performed in a blinded format using a validated computerized image analysis
system (Optimas 5.2). RESULTS: Early (1 month) cardiomyocyte enlargement
decreased to the smallest diameter 6 months posttransplant, but thereafter
progressively increased by 10% to 20% over the subsequent 5- to 6-year
period. Although not statistically established, principal stimuli may
include a discrepancy in body size (recipient > donor), coronary allograft
vasculopathy and posttransplant systemic hypertension. Percent myocardial
fibrosis rose early (1 to 2 months) posttransplant and thereafter remained
at the same modest level of severity. CONCLUSIONS: Cardiomyocyte diameter
of the transplanted heart gradually increases over time, while
percent myocardial fibrosis rises early and remains in a modestly elevated
```

plateau after 2 months posttransplant. These histostructural changes likely contribute to the hemodynamic and cardiac functional alterations commonly observed posttransplant.

Quantitative investigation of cardiomyocyte hypertrophy and myocardial fibrosis over 6 years after **cardiac transplantation**.
... degree and time course over 6 years of cardiomyocyte hypertrophy and myocardial fibrosis of the **cardiac** allograft in **transplanted** patients. BACKGROUND: Diastolic dysfunction and to a certain extent systolic dysfunction are common **cardiac** findings after **heart transplantation**. The **development** of posttransplant cardiomyocyte **hypertrophy** and myocardial fibrosis likely contributes to these derangements. METHODS: Cardiomyocyte diameter and percent fibrosis were determined in serial endomyocardial biopsy specimens obtained from 1 month up to 6 years following **heart transplantation** in 50 patients. Endomyocardial biopsy specimens from 40 patients with primary dilated cardiomyopathy and 11...

... and thereafter remained at the same modest level of severity. CONCLUSIONS: Cardiomyocyte diameter of the **transplanted heart** gradually increases over time, while percent myocardial fibrosis rises early and remains in a modestly...
Descriptors: Cardiomyopathy, Hypertrophic--pathology--PA; *Endomyocardial Fibrosis--pathology--PA; ***Heart Transplantation**--pathology--PA; *Postoperative Complications--pathology--PA

10/3,K,AB/2 (Item 2 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

10964578 97317263 PMID: 9245065
[Acute and chronic heart failure]
Akutni a chronicke srdecni selhani.
Stejfa M; Toman J; Spinarova L
I. interni klinika FN U sv. Anny LF MU, Brno.
Vnitrni lekarstvi (CZECH REPUBLIC) Feb 1997, 43 (2) p105-10, ISSN 0042-773X Journal Code: 0413602
Document type: Journal Article; Review; Review, Tutorial ; English Abstract
Languages: CZECH
Main Citation Owner: NLM
Record type: Completed
Cardiac failure is a syndrome which comprises ventricular dysfunction (confirmed by echocardiography) and compensating mechanisms (immediate activation of the sympathetic nerve and functioning of Starling's mechanism, within hours or days activation of RAAS within days or weeks **hypertrophy** of the heart). Cardiac failure **develops** rapidly either in a previously healthy subject (first extensive IM, diffuse myocarditis, acute aortic or mitral regurgitation) or in a damaged heart (IHD, KMP, defect) as a result of sudden excessive burdening (ischaemia, arrythmia, infection, surgery etc.) or spontaneously (end-stage). It is manifested above all by "backward" failure (pulmonary oedema). The pulmonary pressure must be rapidly reduced: i.v. nitrovasodilators act immediately, i.v. furosemide acts within 10-15 min. (in can, however, reduce the circulating volume which has not increased during the first failure). Also O2, anodynes. In the subacute stage (without any precise time limits) which may develop in serious cases from acute failure, or develop as a result of deterioration of chronic failure, in addition to congestion, symptoms caused by "forward" failure are in the foreground. These are symptoms caused by a reduced minute output and hyperfusion of tissue. It is indicated to administer substances which improve work tolerance, i.e. positive inotropics (digitalis, beta-agonist or phosphodiesterase inhibitors). If the blood pressure drops, a combination

of dopamine and dobutamine should be administered; if the respiratory volume drops, artificial pulmonary ventilation, in case of persisting oedema continuous arteriovenous haemofiltration, in severe failure intraaorrtic balloon contrapulsation etc. In an irreversible state urgent or elective orthoptic **transplantation** of the **heart** should be considered. In chronic heart failure an important component of comprehensive treatment is in addition to treatment of congestion and hypoperfusion, prevention of "cardiovascular remodelling" by means of angiotensin convertase inhibitors etc. Which improve the quality of life and survival. Arrhythmias are an independent prognostic factor.

... of Starling's mechanism, within hours or days activation of RAAS within days or weeks **hypertrophy** of the heart). Cardiac failure **develops** rapidly either in a previously healthy subject (first extensive IM, diffuse myocarditis, acute aortic or...

... in severe failure intraaorrtic balloon contrapulsation etc. In an irreversible state urgent or elective orthoptic **transplantation** of the **heart** should be considered. In chronic heart failure an important component of comprehensive treatment is in...

10/3,K,AB/3 (Item 3 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

10772656 97122733 PMID: 9026850

Hypertensive **cardiac** damage in **heart transplantation**. A noninvasive monitoring study of arterial pressure]

Il danno ipertensivo cardiaco nel trapianto di cuore. Studio con monitoraggio non invasivo della pressione arteriosa.

Cugini P; Chiera A; Petrangeli C M; Capodaglio P F; Voci P; Laurenti A; Papalia U; Marino B; Scibilia G

Semeiotica e Metodologia Medica, Universita La Sapienza, Roma.

Recenti progressi in medicina (ITALY) Oct 1996, 87 (10) p460-5, ISSN 0034-1193 Journal Code: 0401271

Document type: Journal Article ; English Abstract

Languages: ITALIAN

Main Citation Owner: NLM

Record type: Completed

This study is aimed at investigating the relationship between cardiac hypertrophy and blood pressure (BP) 24-h pattern in 34 **heart transplanted** patients (HTP), 9 out of them (26%) being considered as normotensives, the other ones (74%) being regarded as hypertensives under adequate treatment, via casual sphygmomanometry. The study is an attempt to explain the occurrence of at least one sign of hypertrophic cardiopathy in 20 cases (59%), hypothesizing the presence of false normotensives among the putative normotensives and presumably-cured hypertensives. The ambulatory BP monitoring was able to identify 7 hypertensives (78%) among the putative normotensives, and 17 not well-cured subjects (68%) among the presumably cured hypertensives. At least one sign of cardiac hypertrophy was found in 5 (50%) of the 10 true normotensives, who were all non-dipper, and in 15 (63%) of the 24 hypertensives. The 9 hypertensives without cardiac **hypertrophy** (37%) had **developed** hypertension very recently.
These findings stress the role of the ambulatory BP monitoring as a diagnostic tool during the follow-up of HTP, in order to identify the false normotensives as well as the not well-treated hypertensives. This role can contribute to optimize the prophylaxis of hypertensive damage for the **transplanted heart**.

Hypertensive **cardiac** damage in **heart transplantation**. A noninvasive monitoring study of arterial pressure]

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ones (74...

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... treated hypertensives. This role can contribute to optimize the prophylaxis of hypertensive damage for the **transplanted heart**.

Descriptors: Blood Pressure Monitoring, Ambulatory; *Cardiomegaly --diagnosis--DI; ***Heart Transplantation**--physiology--PH; *Hypertension--diagnosis--DI; *Postoperative Complications--diagnosis--DI

10/3,K,AB/4 (Item 4 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

09833881 21644600 PMID: 11784525

[Importance of tumor necrosis factor-alpha in the pathogenesis of heart failure]

Importancia del factor de necrosis tumoral alfa en la patogenia de la insuficiencia cardiaca.

Heberto Herrera Garza Eduardo; Herrera Garza Jose Luis; Rodriguez Gonzalez Humberto; Trevino Trevino Alfonso; Ibarra Flores Marcos; Torre Amione Guillermo

Departamento de Cardiologia del Centro de Medico del Noreste #34 del IMSS, Monterrey NL. Mexico.

Revista espanola de cardiologia (Spain) Jan 2002, 55 (1) p61-6, ISSN 0300-8932 Journal Code: 0404277

Document type: Journal Article; Review; Review, Tutorial ; English Abstract

Languages: SPANISH

Main Citation Owner: NLM

Record type: Completed

Clinical and experimental evidence demonstrating the effects of tumor necrosis factor-alpha (TNF-alpha) in patients with heart failure continues to accumulate. It is well established that high concentrations of TNF-alpha appear in the circulation of patients with heart failure and that these levels have a directly proportional correlation with the patient's functional class. TNF-alpha levels also show a linear relation with prognosis. These circulating levels are responsible for the decreased expression of myocardial TNF-alpha receptors observed in heart failure. As a result of extrapolation of findings from experimental animals, we assume that TNF-alpha is deleterious to myocardial function in humans because it induces a negative inotropic state in patients who have not undergone **heart transplant** . Supporting this assumption is the fact that the resolution or improvement of pressure overload (obstructive hypertrophic myocardiopathy, by ethanol ablation) and volume overload (terminal dilated myocardiopathy, by ventricular assistance) states is accompanied by a decrease in myocardial TNF-alpha expression.The use of specific antagonists of circulating TNF-alpha in patients with symptomatic heart failure has been demonstrated to be safe and possibly effective. At present, multicenter studies are under way to assess the efficacy of this antagonism in a larger number of patients. If the results of these studies are favorable, we will have new therapeutic elements for managing patients with advanced hear failure.The **transplanted heart** behaves differently from the native heart. From the early stages of HTx, myocardial TNF-alpha expression is greatly increased (much more than in patients with heart failure) and not associated with contractile dysfunction, in contrast with what occurs in the native **heart**. However, we know that the **transplanted heart** soon **develops** ventricular **hypertrophy**, fibrosis, diastolic dysfunction, and late graft failure, even in the presence of normal epicardial coronary arteries. Clinical

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; Heart Failure, Congestive--drug therapy--DT; **Heart** Failure, Congestive--surgery--SU; **Heart Transplantation**; Myocardium --metabolism--ME; Receptors, Tumor Necrosis Factor--physiology--PH; Tumor Necrosis Factor--antagonists and inhibitors...

10/3,K,AB/5 (Item 5 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

6/30

09597496 21381694 PMID: 11489774
Cardiac hypertrophy after **transplantation** is associated with persistent expression of tumor necrosis factor-alpha.
Stetson S J; Perez-Verdia A; Mazur W; Farmer J A; Koerner M M; Weilbaecher D G; Entman M L; Quinones M A; Noon G P; Torre-Amione G
Department of Medicine, The DeBakey Heart Center, The Winters Center for Heart Failure Research, Baylor College of Medicine, Houston, Texas, USA.
Circulation (United States) Aug 7 2001, 104 (6) p676-81, ISSN 1524-4539 Journal Code: 0147763
Contract/Grant No.: HL-42550; HL; NHLBI
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
BACKGROUND: The mechanisms that contribute to cardiac allograft hypertrophy are not known; however, the rapid progression and severity of hypertrophy suggest that nonhemodynamic factors may play a contributory role. Tumor necrosis factor-alpha (TNF-alpha) is a cytokine produced in cardiac allografts and capable of producing hypertrophy and fibrosis; therefore, we suggest that TNF-alpha may play a contributory role. Accordingly, the aims of our study were to define the role of systemic hypertension in the **development** of **hypertrophy**, characterize the histological determinants of hypertrophy, and characterize the expression of myocardial TNF-alpha after **heart transplantation**.
METHODS AND RESULTS: To separate the effect of hypertension from immune injury in the **development** of cardiac allograft **hypertrophy**, we measured the gain in left ventricular mass by 2D echocardiography in **heart transplant** recipients and lung **transplant** recipients who developed similar rates of systemic hypertension. The gain in left ventricular mass was 73% in **heart transplant** recipients and 7% in lung transplant recipients (P<0.0001). By comparing myocardial samples obtained during the first week after transplant and at 1 year, we found that there was a significant increase in total collagen content (P<0.0001), collagen I (P<0.0001), collagen III (P<0.0001), and myocyte size (P<0.0001). These changes were associated with persistent myocardial TNF-alpha expression. CONCLUSIONS: We suggest that the contribution of hypertension to cardiac allograft hypertrophy is minimal and that

persistent intracardiac expression of TNF-alpha may contribute to the **development** of cardiac allograft **hypertrophy**.

Cardiac hypertrophy after **transplantation** is associated with persistent expression of tumor necrosis factor-alpha.
...the aims of our study were to define the role of systemic hypertension in the **development** of **hypertrophy** , characterize the histological determinants of hypertrophy, and characterize the expression of myocardial TNF-alpha after **heart transplantation**. METHODS AND RESULTS: To separate the effect of hypertension from immune injury in the **development** of cardiac allograft **hypertrophy**, we measured the gain in left ventricular mass by 2D echocardiography in **heart transplant** recipients and lung **transplant** recipients who developed similar rates of systemic hypertension. The gain in left ventricular mass was 73% in **heart transplant** recipients and 7% in lung transplant recipients (P<0.0001). By comparing myocardial samples obtained...

... hypertrophy is minimal and that persistent intracardiac expression of TNF-alpha may contribute to the **development** of cardiac allograft **hypertrophy**.
Descriptors: Cardiomegaly--metabolism--ME; ***Heart Transplantatio n**; *Tumor Necrosis Factor--biosynthesis--BI

10/3,K,AB/6 (Item 6 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

09445153 21216994 PMID: 11316525
Assessment of the myocardial changes in **heart transplant** recipients without evident acute myocardial rejection by integrated backscatter: comparison with simultaneous dobutamine stress echocardiography and (201)thallium spect.
Ho Y L; Chen C L; Hsu R B; Lin L C; Yen R F; Lee C M; Chen M F; Huang P J
Department of Internal Medicine (Cardiology), National Taiwan University Hospital, No. 7, Chung-Shan S. Road, Taipei, Taiwan.
Ultrasound in medicine & biology (England) Feb 2001, 27 (2) p171-9, ISSN 0301-5629 Journal Code: 0410553
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Cardiomyocyte **hypertrophy** and interstitial fibrin deposition **develop** in cardiac allografts and contribute to the functional changes of transplanted hearts. We hypothesized that integrated backscatter (IBS) can detect these myocardial changes. A total of 32 **heart transplant** recipients with either no or mild acute rejection (International Society of **Heart** and Lung **Transplantation** grade IA) were enrolled in this study. IBS data of myocardium were collected immediately before simultaneous dobutamine stress echocardiography (DSE) and (201)thallium imaging. Coronary angiography and endomyocardial biopsy were also performed. Coronary angiography showed diffuse narrowing in 1 patient who also had abnormal results of IBS, DSE, and thallium results. In the other 31 patients with patent coronary arteries, there were 3 patients (10%) with abnormal DSE results, 19 patients (61%) with abnormal IBS patterns, and 16 patients (52%) with reversible thallium perfusion defects. Of the patients, 44% had cardiomyocyte hypertrophy and 56% interstitial fibrin deposition. There were significant differences in the prevalence of (201)thallium perfusion defects and serum cyclosporine levels between patients with and without abnormal IBS patterns. Pathologic changes were also associated with abnormal IBS patterns (p = 0.01). However, there was no association between abnormal IBS and DSE results. By multiple logistic regression analysis, the abnormal IBS patterns were associated inversely

with serum cyclosporine level (p = 0.028). In conclusion, abnormal IBS patterns are associated significantly with perfusion heterogeneity and pathologic changes in **heart transplant** recipients without evident acute myocardial rejection. There is no association between abnormal IBS patterns and dobutamine-induced dyssynergy in these patients. IBS provides a noninvasive approach for detection of myocardial changes in transplanted hearts without evident acute rejection.

Assessment of the myocardial changes in **heart transplant** recipients without evident acute myocardial rejection by integrated backscatter: comparison with simultaneous dobutamine stress echocardiography...

Cardiomyocyte **hypertrophy** and interstitial fibrin deposition **develop** in cardiac allografts and contribute to the functional changes of transplanted hearts. We hypothesized that integrated backscatter (IBS) can detect these myocardial changes. A total of 32 **heart transplant** recipients with either no or mild acute rejection (International Society of **Heart** and Lung **Transplantation** grade IA) were enrolled in this study. IBS data of myocardium were collected immediately before...

... In conclusion, abnormal IBS patterns are associated significantly with perfusion heterogeneity and pathologic changes in **heart transplant** recipients without evident acute myocardial rejection. There is no association between abnormal IBS patterns and...

Descriptors: Coronary Disease--ultrasonography--US; ***Heart Transplantation**...; Disease--physiopathology--PP; Coronary Disease --radionuclide imaging--RI; Dobutamine--diagnostic use--DU; Echocardiography; Exercise Test; **Heart** Catheterization; **Heart Transplantation**--adverse effects--AE; Logistic Models; Middle Age; Observer Variation; Thallium Radioisotopes--diagnostic use--DU; Tomography ...

10/3,K,AB/7 (Item 7 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

09084822 20382028 PMID: 10928294

Cardiac and vascular abnormalities in renal **transplant** patients: differential effects of cyclosporin and azathioprine.

Galiatsou E; Morris S T; Jardine A G; Rodger R S; Watson M A; Elliott H L
Department of Medicine and Therapeutics, Western Infirmary, Glasgow, UK.

Journal of nephrology (ITALY) May-Jun 2000, 13 (3) p185-92, ISSN 1120-3625 Journal Code: 9012268
Document type: Clinical Trial; Controlled Clinical Trial; Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed

Renal transplant patients die prematurely of cardiovascular disease and LV hypertrophy is now recognised as an important adverse prognostic indicator. This study investigated the factors implicated in the **development** of echocardiographic abnormalities (including LV **hypertrophy**) and the possible differential effects of treatment with cyclosporin and azathioprine. A cross-sectional study was undertaken in 46 patients randomly assigned to immunosuppressant treatment with either cyclosporin or azathioprine at 1 year post-transplantation: patients were studied not less than 5 years after assignment to cyclosporin (CyA) - or azathioprine (Aza)-based treatment regimens. Although clinic blood pressure control was not different in the two treatment groups, 24 hour ambulatory BP (ABP), particularly night-time BP, was significantly higher in the CyA group. There was a trend for both left ventricular hypertrophy (61 vs. 43%) and carotid wall thickening (43 vs. 26%) to be more common in the CyA group though this failed to achieve statistical significance. Left ventricular mass was determined by ABP, rather than clinic BP, and was also associated

with increased QT dispersion. Multivariate analysis identified that 24 hour ambulatory systolic blood pressure (ASBP) and time on renal replacement therapy (RRT) were the major determinants of LV mass. Thus, despite the absence of differences in clinic BP measurements, CyA treatment was associated with higher rates of cardiovascular functional and structural abnormalities. This small scale study has identified cardiovascular functional and structural abnormalities in renal transplant patients, particularly in those receiving CyA-based immunosuppressive therapy. However, rather than reflecting a direct effect of CyA they are related to increased 24 ABP (but not clinic BP). These data suggest that ABP should be used to monitor and target antihypertensive therapy in this high risk patient group. Moreover, the future use of non-calcineurin inhibitor immunosuppressant therapy may have benefits on blood pressure control and LV mass.

Cardiac and vascular abnormalities in renal **transplant** patients: differential effects of cyclosporin and azathioprine.

... recognised as an important adverse prognostic indicator. This study investigated the factors implicated in the **development** of echocardiographic abnormalities (including LV **hypertrophy**) and the possible differential effects of treatment with cyclosporin and azathioprine. A cross-sectional study...

10/3,K,AB/8 (Item 8 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

08924493 20212810 PMID: 10750772

Arterial switch after failed atrial baffle procedures for transposition of the great arteries.

Mavroudis C; Backer C L

Children's Memorial Hospital and Department of Surgery, Northwestern University Medical School, Chicago, Illinois 60614-3394, USA. c-mavroudis@nwu.edu

Annals of thoracic surgery (UNITED STATES) Mar 2000, 69 (3) p851-7, ISSN 0003-4975 Journal Code: 15030100R

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

BACKGROUND: Late failure of the systemic right ventricle after atrial baffle procedures in patients with transposition of the great arteries poses significant management problems. We reviewed our experience with staged conversion to arterial switch operation (ASO) in these patients. METHODS: Between 1984 and 1999, 11 patients underwent pulmonary artery band (PAB) to prepare the left ventricle for ASO conversion. One additional patient had subpulmonic stenosis and was naturally prepared. Mean age at the initial PAB was 12.2+/-7 years (range, 1.9 to 23 years). Four patients underwent reoperation to tighten the PAB before ASO. Mean interval from PAB to ASO was 1.3+/-0.9 years. RESULTS: There was no mortality from PAB. Six patients had ASO conversion and 2 died. Recent surgical modifications at the time of ASO were used to prevent neoaortic valve insufficiency and to cryoablate atrial reentry tachycardia. Four patients developed biventricular failure after PAB and had orthotopic **cardiac transplantation** (OCT) 14+/-10 months after PAB. The other 2 patients are still with PAB: 1 is awaiting ASO conversion and the other has insufficient left ventricular hypertrophy necessary for ASO conversion despite two preparatory PABs. CONCLUSIONS: A select group of patients with right ventricular failure after atrial baffle operations can undergo staged conversion to ASO with the opportunity for excellent long-term outcome. Surgical modifications at the time of ASO can address the problems of neoaortic insufficiency and persistent atrial arrhythmias. PAB may be a therapeutic endpoint in some patients not responding with adequate left

ventricular **hypertrophy**. Those patients who **develop** biventricular failure after PAB will require **cardiac transplantation**.

... to cryoablate atrial reentry tachycardia. Four patients developed biventricular failure after PAB and had orthotopic **cardiac transplantation** (OCT) 14+/-10 months after PAB. The other 2 patients are still with PAB: 1...

... PAB may be a therapeutic endpoint in some patients not responding with adequate left ventricular **hypertrophy**. Those patients who **develop** biventricular failure after PAB will require **cardiac transplantation**.

10/3,K,AB/9 (Item 9 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

6/30

08743685 20023767 PMID: 10561111

Influence of felodipine on left ventricular hypertrophy and systolic function in orthotopic **heart transplant** recipients: possible interaction with cyclosporine medication.

Schwitter J; De Marco T; Globits S; Sakuma H; Klinski C; Chatterjee K; Parmley W W; Higgins C B

Department of Radiology, University of California, San Francisco 94143-0628, USA.

Journal of heart and lung transplantation - the official publication of the International Society for Heart Transplantation (UNITED STATES) Oct 1999, 18 (10) p1003-13, ISSN 1053-2498 Journal Code: 9102703

Document type: Clinical Trial; Journal Article; Randomized Controlled Trial

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

BACKGROUND: Concentric left ventricular (LV) **hypertrophy develops** early in orthotopic **heart transplant** (OHT) recipients. To compare the effects of a calcium channel blocker, felodipine, versus diuretics on LV hypertrophy and LV systolic function repeated magnetic resonance imaging studies were performed in OHT recipients. Cyclosporine levels and neurohormones were also measured to explore potential interactions with treatment. METHODS: Twenty-two patients were randomized at baseline (2 months after OHT) to receive felodipine or diuretic treatment. Before and after 4 months of treatment (n = 19), LV dimensions and LV mass (Simpson's rule) were measured. The relationship between circumferential fiber shortening (two-shell cylindrical model) and end-systolic wall stress was used as a measure of load-independent LV contractility. Neurohormones were measured at the beginning and end of the treatment period, and cyclosporine levels and blood pressures were additionally measured during treatment. RESULTS: At baseline, the felodipine and diuretic groups did not differ in LV mass, wall stress, and fiber shortening. During felodipine treatment LV mass decreased ($p < 0.01$) and tended to increase during diuretics treatment ($p = 0.06$). Afterload-corrected fiber shortening did not change during felodipine treatment, but decreased ($p < 0.01$) with diuretics. Changes in LV mass were positively correlated with cyclosporine levels ($r = 0.70$) in the diuretics group, but not in the felodipine group. CONCLUSIONS: In OHT recipients during diuretic treatment, progression of LV hypertrophy occurs in relation to cyclosporine plasma levels and is accompanied by impairment of systolic contractile function. Felodipine induces regression of LV hypertrophy, while systolic contractile function is preserved. During felodipine treatment, regression of LV hypertrophy is unrelated to cyclosporine levels. Thus, felodipine seems to attenuate the hypertrophic effect of cyclosporine on transplanted hearts.

Influence of felodipine on left ventricular hypertrophy and systolic function in orthotopic **heart transplant** recipients: possible interaction with cyclosporine medication.

BACKGROUND: Concentric left ventricular (LV) **hypertrophy d velops** early in orthotopic **heart transplant** (OHT) recipients. To compare the effects of a calcium channel blocker, felodipine, versus diuretics on...

Descriptors: Antihypertensive Agents--therapeutic use--TU; *Cyclosporine --therapeutic use--TU; *Felodipine--therapeutic use--TU; ***Heart Transplantation**--physiology--PH; *Hypertrophy, Left Ventricular--drug therapy--DT; *Immunosuppressive Agents--therapeutic use--TU; Analysis of Variance; Diuretics--therapeutic use--TU; Drug Interactions; **Heart Transplantation**--pathology--PA; **Heart Transplantation** --statistics and numerical data--SN; **Heart** Ventricle--drug effects --DE; Heart Ventricle--pathology--PA; Heart Ventricle--physiopathology --PP; Hypertrophy, Left Ventricular...

10/3,K,AB/10 (Item 10 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

08635515 95324042 PMID: 7600648

Prerequisite for cardiac aldosterone action. Mineralocorticoid receptor and 11 beta-hydroxysteroid dehydrogenase in the human heart.

Lombes M; Alfaidy N; Eugene E; Lessana A; Farman N; Bonvalet J P

INSERM U246, Institut Federatif de Recherche Cellules epitheliales, Faculte de Medecine X, Bichat, Paris, France.

Circulation (UNITED STATES) Jul 15 1995, 92 (2) p175-82, ISSN 0009-7322 Journal Code: 0147763

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

BACKGROUND: It has been proposed that aldosterone exerts direct effects on heart function, most notably on the **development** of myocardial fibrosis during ventricular **hypertrophy** in rat. Initial events in aldosterone action entail its binding to mineralocorticoid receptor (MR). Because MR displays similar affinities for aldosterone and glucocorticoids, the in vivo aldosterone selectivity of MR requires the presence of an enzyme, 11 beta-hydroxysteroid dehydrogenase (11-HSD), which metabolizes glucocorticoids into inactive derivatives. Although evidence exists for the presence of MR in rodent heart, no data are available for humans; moreover, the existence of cardiac 11-HSD is controversial. METHODS AND RESULTS: The heart samples used originated from tissue removed during cardiac surgery in nontransplant patients or from endocavitary biopsies done for the follow-up of **heart transplantation**. The expression of MR was examined at the mRNA and protein level by in situ hybridization with cRNA probes specific for human MR mRNA and by immunodetection with two specific anti-MR antibodies. 11-HSD catalytic activity was determined by measurement of the metabolic rate of tritiated corticosteroids by cardiac samples. In nontransplanted hearts, an in situ hybridization signal equivalent to that found in the whole kidney was present on cardiomyocytes. Specific immunolabeling of cardiomyocytes with anti-MR antibodies demonstrated the presence of the MR protein. Cardiac 11-HSD activity was detected (243 +/- 26 fmol.30 min-1.mg protein-1) and was dependent on the cofactor NAD, not NADP, suggesting that it corresponds to the form of the enzyme specifically responsible for MR protection. In transplanted hearts that presented severe alterations, MR immunodetection was weaker and irregular, with no specific hybridization signal. CONCLUSIONS: Our results demonstrate that MR is coexpressed with 11-HSD in human heart, which thus possesses the cellular machinery required for direct aldosterone action.

... has been proposed that aldosterone exerts direct effects on heart function, most notably on the **development** of myocardial fibrosis during ventricular **hypertrophy** in rat. Initial events in aldosterone action entail its binding to mineralocorticoid receptor (MR). Because...

... cardiac surgery in nontransplant patients or from endocavitary biopsies done for the follow-up of **heart transplantation**. The expression of MR was examined at the mRNA and protein level by in situ...

10/3,K,AB/11 (Item 11 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

6/30

08043274 94109009 PMID: 8281642

Structural basis of end-stage failure in ischemic cardiomyopathy in humans.

Beltrami C A; Finato N; Rocco M; Feruglio G A; Puricelli C; Cigola E; Quaini F; Sonnenblick E H; Olivetti G; Anversa P

Department of Pathology, University of Udine, Italy.

Circulation (UNITED STATES) Jan 1994, 89 (1) p151-63, ISSN 0009-7322 Journal Code: 0147763

Contract/Grant No.: HL-38132; HL; NHLBI; HL-39901; HL; NHLBI; HL-40561; HL; NHLBI; +

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

BACKGROUND: Ischemic cardiomyopathy is characterized by myocyte loss, reactive cellular hypertrophy, and ventricular scarring. However, the relative contribution of these tissue and cellular processes to late failure remains to be determined. METHODS AND RESULTS: Ten hearts were obtained from individuals undergoing **cardiac transplantation** as a result of chronic coronary artery disease in its terminal stage. An identical number of control hearts were collected at autopsy from patients who died from causes other than cardiovascular disease, and morphometric methodologies were applied to the analysis of the left and right ventricular myocardium. Left ventricular hypertrophy evaluated as a change in organ weight, aggregate myocyte mass, and myocyte cell volume per nucleus showed increases of 85%, 47%, and 103%, respectively. Corresponding increases in the right ventricle were 75%, 74%, and 112%. Myocyte loss, which accounted for 28% and 30% in the left and right ventricles, was responsible for the difference in the assessment of myocyte hypertrophy at the ventricular, tissue, and cellular levels. Left ventricular muscle cell hypertrophy was accomplished through a 16% and 51% increase in myocyte diameter and length, whereas right ventricular myocyte hypertrophy was the consequence of a 13% and 67% increase in these linear dimensions, respectively. Moreover, a 36% reduction in the number of myocytes included in the thickness of the left ventricular wall was found. Collagen accumulation in the form of segmental, replacement, and interstitial fibrosis comprised an average 28% and 13% of the left and right ventricular myocardia, respectively. The combination of cell loss and myocardial fibrosis, myocyte lengthening, and mural slippage of cells resulted in 4.6-fold expansion of left ventricular cavitary volume and a 56% reduction in the ventricular mass-to-chamber volume ratio. CONCLUSIONS: These results are consistent with the contention that both myocyte and collagen compartments participate in the **development** of decompensated eccentric ventricular **hypertrophy** in the cardiomyopathic heart of ischemic origin.

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... results are consistent with the contention that both myocyte and collagen compartments participate in the **development** of decompensated eccentric ventricular **hypertrophy** in the cardiomyopathic heart of ischemic origin.
...; Congestive--pathology--PA; Coronary Arteriosclerosis--pathology--PA; Coronary Vessels--pathology--PA; Endomyocardial Fibrosis--pathology--PA; **Heart Transplantation**; Hypertrophy, Left Ventricular--pathology --PA; Middle Age; Organ Weight

10/3,K,AB/12 (Item 12 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

07496919 92360622 PMID: 1386753
Circadian rhythms of heart rate and blood pressure after **heart transplantation**.
Dart A M; Yeoh J K; Jennings G L; Cameron J D; Esmore D S
Alfred and Baker Medical Unit, Alfred Hospital, Prahran, Victoria, Australia.
Journal of heart and lung transplantation - the official publication of the International Society for Heart Transplantation (UNITED STATES) Jul-Aug 1992, 11 (4 Pt 1) p784-92, ISSN 1053-2498 Journal Code: 9102703
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Blood pressure and heart rate were recorded over 24-hour periods on 39 occasions in 20 subjects 5 to 72 weeks after **heart transplantation** . All patients were receiving cyclosporine, azathioprine, and prednisolone. In 38 of the 39 records the mean nighttime heart rate was lower than the mean daytime rate, with a peak difference of 20.1 +/- 1.8 beats/min. Blood pressure responses were, however, of two patterns. In 15 of the 39 recordings (approximately 50% of patients) the mean nighttime systolic pressure was higher than the mean daytime systolic pressure; in the remainder the converse was observed. The pattern was generally consistent on repeated recordings from the same patient and was not related to time since transplantation, renal function, or other therapy. Echocardiographic/Doppler studies were available at the time of 31 of these recordings. No differences in left ventricular diameters, systolic function, or transmitral filling patterns were present between patients whose blood pressure was higher or lower at night. Left ventricular posterior wall thickness and the ratio between wall thickness and ventricular diameter at end diastole were greater in the group showing nighttime pressure falls. Blood pressure responses after **heart transplantation** show the presence of nighttime "dippers" and "nondippers." At least early after transplantation, however, nondipper status is not preferentially associated with the **development** of left ventricular **hypertrophy**. The mechanisms accounting for the different circadian blood pressure responses in **heart transplant** recipients are not known.

Circadian rhythms of heart rate and blood pressure after **heart transplantation**.
... over 24-hour periods on 39 occasions in 20 subjects 5 to 72 weeks after **heart transplantation** . All patients were receiving cyclosporine, azathioprine, and prednisolone. In 38 of the 39 records the ...

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transplantation, however, nondipper status is not preferentially associated with the **development** of left ventricular **hypertrophy**. The mechanisms accounting for the different circadian blood pressure responses in **heart transplant** recipients are not known.
Descriptors: Blood Pressure--physiology--PH; *Circadian Rhythm --physiology--PH; ***Heart** Rate--physiology--PH; ***Heart Transplantation**--physiology--PH

10/3,K,AB/13 (Item 13 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

07163612 92025819 PMID: 1833962
Blood pressure and left ventricular anatomy and function after **heart transplantation**.
Leenen F H; Holliwell D L; Cardella C J
Department of Medicine, Toronto Western Hospital, Canada.
American heart journal (UNITED STATES) Oct 1991, 122 (4 Pt 1) p1087-94, ISSN 0002-8703 Journal Code: 0370465
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
To determine whether hypertension occurring after **heart** transplant causes the **development** of **cardiac hypertrophy**, changes in pressure load (N = 13) and left ventricular anatomy (N = 11) were evaluated up to 1 year after **heart transplant** in a prospective longitudinal study. Pressure load was evaluated by 24-hour ambulatory blood pressure monitoring, and left ventricular anatomy and function were assessed by M-mode echocardiography under two-dimensional guidance. Body weight increased by 11 to 12 kg. Blood pressure showed a gradual increase during the first few months after transplant: diastolic pressure by 15 to 18 mm Hg and systolic pressure by 12 to 15 mm Hg, with hypertension persisting during the night. Nearly all patients required treatment with one or two antihypertensive drugs. The increase in blood pressure was related to increased total peripheral resistance with minor decreases in cardiac output. Both septal and posterior wall thickness and left ventricular mass (by 25 to 30 gm/m2) decreased during the initial months after transplant and subsequently remained at "normal" levels (100 gm/m2). The persistence of normal left ventricular mass may indicate either that the increases in daily pressure load and body weight were not sufficient to induce myocardial growth or that the latter was prevented by, for example, absence of cardiac sympathetic nerve activity.

Blood pressure and left ventricular anatomy and function after **heart transplantation**.
To determine whether hypertension occurring after **heart** transplant causes the **development** of **cardiac hypertrophy**, changes in pressure load (N = 13) and left ventricular anatomy (N = 11) were evaluated up to 1 year after **heart transplant** in a prospective longitudinal study. Pressure load was evaluated by 24-hour ambulatory blood pressure...
Descriptors: Cardiomegaly--etiology--ET; ***Heart Transplantation%%--pathology--PA**; *Heart Transplantation--**physiology--PH**; ***Hypertension--complications--CO**; ***Postoperative Complications--physiopathology--PP**

10/3,K,AB/14 (Item 14 from file: 155)
DIALOG(R)File 155:MEDLINE(R)
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05362974 87041192 PMID: 3774698

The morphological progression of viral myocarditis.
Billingham M E; Tazelaar H D
Postgraduate medical journal (ENGLAND) Jun 1986, 62 (728) p581-4,
ISSN 0032-5473 Journal Code: 0234135
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
In an attempt to document the morphological progression from acute idiopathic myocarditis to end-stage dilated cardiomyopathy we studied 20 patients with a diagnosis of myocarditis who had had serial endomyocardial biopsies performed with intervals of 1 month to 2 years and whose ages varied from 6 months to 62 years. Fifteen of these patients were treated with immunosuppressive drugs for myocarditis. Ten out of 15 treated patients stabilized clinically. In the remaining 5 cases there was worsening congestive heart failure and 1 patient underwent cardiac transplantation . Of the 5 patients who did not receive immunosuppression, 2 stabilized spontaneously, and 3 developed heart failure, 2 of whom subsequently had cardiac transplants. Whether the patients received immunosuppression or not, in all cases, the inflammatory infiltrate was less but the myocardium developed significant hypertrophy with an increase in interstitial fibrosis and in 8 cases the morphological changes were those of dilated cardiomyopathy. From the morphological standpoint of this study we have shown some evidence that dilated cardiomyopathy can be the end result of acute myocarditis. It appears that not every case of acute myocarditis progresses to dilated cardiomyopathy and that steroid treatment does not necessarily prevent progression of myocarditis to dilated cardiomyopathy.

... In the remaining 5 cases there was worsening congestive heart failure and 1 patient underwent cardiac transplantation . Of the 5 patients who did not receive immunosuppression, 2 stabilized spontaneously, and 3 developed heart failure, 2 of whom subsequently had cardiac transplants . Whether the patients received immunosuppression or not, in all cases, the inflammatory infiltrate was less but the myocardium developed significant hypertrophy with an increase in interstitial fibrosis and in 8 cases the morphological changes were those ...

10/3,K,AB/15 (Item 1 from file: 55)
DIALOG(R)File 55:Biosis Previews(R)

12898095 BIOSIS NO.: 200100105244
Contribution of non-hemodynamic factors to development of hypertrophy after heart transplantation: A potential role for tumor necrosis factor-alpha.
AUTHOR: Perez-Verdia Alex(a); Stetson Sonny J(a); Mazur Wojciech(a); Quinones Miguel A; Entman Mark L; Noon George P; Torre-Amione Guillermo
AUTHOR ADDRESS: (a)Baylor Coll of Medicine, Houston, TX**USA
JOURNAL: Circulation 102 (18 Supplement):pII504 October 31, 2000
MEDIUM: print
CONFERENCE/MEETING: Abstracts from Scientific Sessions 2000 New Orleans, Louisiana, USA November 12-15, 2000
ISSN: 0009-7322
RECORD TYPE: Citation
LANGUAGE: English
SUMMARY LANGUAGE: English
2000

Contribution of non-hemodynamic factors to development of hypertrophy after heart transplantation: A potential role for tumor necrosis factor-alpha.

DESCRIPTORS:
...DISEASES: heart disease, transplantation treatment
METHODS & EQUIPMENT: heart transplantation--

10/3,K,AB/16 (Item 2 from file: 55)
DIALOG(R)File 55:Biosis Previews(R)

11662844 BIOSIS NO.: 199800444575
Quantitative investigation of cardiomyocyte hypertrophy and myocardial fibrosis over 6 years cardiac transplantation.
AUTHOR: Armstrong Arthur T; Binkley Philip F; Baker Peter B; Myerowitz P David; Leier Carl V(a)
AUTHOR ADDRESS: (a)Div. Cardiol., Ohio State Univ. Hosp., 669 Means Hall, 1654 Upham Dr., Columbus, OH 43210**USA
JOURNAL: Journal of the American College of Cardiology 32 (3):p704-710 Sept., 1998
ISSN: 0735-1097
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Objectives. This study was performed to determine the degree and time course over 6 years of cardiomyocyte hypertrophy and myocardial fibrosis of the cardiac allograft in transplanted patients. Background. Diastolic dysfunction and to a certain extent systolic dysfunction are common cardiac findings after heart transplantation. The development of posttransplant cardiomyocyte hypertrophy and myocardial fibrosis likely contributes to these derangements. Methods. Cardiomyocyte diameter and percent fibrosis were determined in serial endomyocardial biopsy specimens obtained from I month up to 6 years following heart transplantation in 50 patients. Endomyocardial biopsy specimens from 40 patients with primary dilated cardiomyopathy and 11 normal subjects were similarly analyzed for control data. Analyses were performed in a blinded format using a validated computerized image analysis system (Optimas 5.2). Results. Early (1 month) cardiomyocyte enlargement decreased to the smallest diameter 6 months posttransplant, but thereafter progressively increased by 10% to 20% over the subsequent 5- to 6-year period. Although not statistically established, principal stimuli may include a discrepancy in body size (recipient > donor), coronary allograft vasculopathy and posttransplant systemic hypertension. Percent myocardial fibrosis rose early (1 to 2 months) posttransplant and thereafter remained at the same modest level of severity. Conclusions. Cardiomyocyte diameter of the transplanted heart gradually increases over time, while percent myocardial fibrosis rises early and remains in a modestly elevated plateau after 2 months posttransplant. These histostructural changes likely contribute to the hemodynamic and cardiac functional alterations commonly observed posttransplant.

1998
Quantitative investigation of cardiomyocyte hypertrophy and myocardial fibrosis over 6 years cardiac transplantation.

...ABSTRACT: degree and time course over 6 years of cardiomyocyte hypertrophy and myocardial fibrosis of the cardiac allograft in transplanted patients. Background. Diastolic dysfunction and to a certain extent systolic dysfunction are common cardiac findings after heart transplantation. The development of posttransplant cardiomyocyte hypertrophy and myocardial fibrosis likely contributes to these derangements. Methods. Cardiomyocyte diameter and percent fibrosis were determined in serial endomyocardial biopsy specimens obtained from I month up to 6 years following heart

transplantation in 50 patients. Endomyocardial biopsy specimens from 40 patients with primary dilated cardiomyopathy and 11...

...and thereafter remained at the same modest level of severity. Conclusions. Cardiomyocyte diameter of the transplanted heart gradually increases over time, while percent myocardial fibrosis rises early and remains in a modestly...
METHODS & EQUIPMENT: cardiac transplantation--

10/3,K,AB/17 (Item 1 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

10355704 Genuine Article#: 516FE Number of References: 24
Title: Importance of tumor necrosis factor-alpha in the pathogenesis of heart failure (ABSTRACT AVAILABLE)
Author(s): Garza EHH; Garza JLH; Gonzalez HR; Trevino AT; Flores MI; Amione GT (REPRINT)
Corporate Source: 6550 Fannin,MS SM 1901/Houston//TX/77030 (REPRINT); IMSS,Dept Cardiol, Ctr Med Noreste 34,Monterrey/NL/Mexico/; IMSS,Dept Cirugia Cardiovasc & Torac, Ctr Med Noreste 34,Monterrey/NL/Mexico/; Hosp Univ Dr Jose Eleuterio Gonzalez,Dept Hematol,Monterrey/NL/Mexico/; Baylor Coll Med,Dept Med, Secc Cardiol, Winters Ctr Heart Failure Res,Houston//TX/77030; Methodist Hosp,Houston//TX/77030
Journal: REVISTA ESPANOLA DE CARDIOLOGIA, 2002, V55, N1 (JAN), P61-66
ISSN: 0300-8932 Publication date: 20020100
Publisher: EDICIONES DOYMA S/L, TRAV DE GRACIA 17-21, 08021 BARCELONA, SPAIN
Language: Spanish Document Type: ARTICLE
Abstract: Clinical and experimental evidence demonstrating the effects of tumor necrosis factor-alpha (TNF-alpha) in patients with heart failure continues to accumulate. It is well established that high concentrations of TNF-alpha appear in the circulation of patients with heart failure and that these levels have a directly proportional correlation with the patient's functional class. TNF-alpha levels also show a linear relation with prognosis. These circulating levels are responsible for the decreased expression of myocardial TNF-alpha receptors observed in heart failure.

As a result of extrapolation of findings from experimental animals, we assume that TNF-alpha is deleterious to myocardial function in humans because it induces a negative inotropic state in patients who have not undergone heart transplant. Supporting this assumption is the fact that the resolution or improvement of pressure overload (obstructive hypertrophic myocardiopathy, by ethanol ablation) and volume overload (terminal dilated myocardiopathy, by ventricular assistance) states is accompanied by a decrease in myocardial TNF-alpha expression.

The use of specific antagonists of circulating TNF-alpha in patients with symptomatic heart failure has been demonstrated to be safe and possibly effective. At present, multicenter studies are under way to assess the efficacy of this antagonism in a larger number of patients. If the results of these studies are favorable, we will have new therapeutic elements for managing patients with advanced hear failure.

The transplanted heart behaves differently from the native heart. From the early stages of HTx, myocardial TNF-alpha expression is greatly increased (much more than in patients with heart failure) and not associated with contractile dysfunction, in contrast with what occurs in the native heart. However, we know that the transplanted heart soon develops ventricular hypertrophy, fibrosis, diastolic dysfunction, and late graft

failure, even in the presence of normal epicardial coronary arteries. Clinical evidence suggests that TNF-alpha may be involved in these processes.

...Abstract: in humans because it induces a negative inotropic state in patients who have not undergone heart transplant. Supporting this assumption is the fact that the resolution or improvement of pressure overload (obstructive...

...favorable, we will have new therapeutic elements for managing patients with advanced hear failure.

The transplanted heart behaves differently from the native heart. From the early stages of HTx, myocardial TNF-alpha...
...failure) and not associated with contractile dysfunction, in contrast with what occurs in the native heart. However, we know that the transplanted heart soon develops ventricular hypertrophy, fibrosis, diastolic dysfunction, and late graft failure, even in the presence of normal epicardial coronary...

10/3,K,AB/18 (Item 2 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

10225527 Genuine Article#: 497YP Number of References: 101
Title: Importance of training-induced effects on the arterial vascular system and the skeletal musculature in the therapy regime of congestive heart failure NYHA II/III (ABSTRACT AVAILABLE)
Author(s): Huonker M (REPRINT) ; Keul J
Corporate Source: Univ Freiburg,Med Klin, Abt Pravent Rehabil & SportMed,Hugstetter Str 55/D-79106 Freiburg//Germany/ (REPRINT); Univ Freiburg,Med Klin, Abt Pravent Rehabil & SportMed,D-79106 Freiburg//Germany/
Journal: ZEITSCHRIFT FUR KARDIOLOGIE, 2001, V90, N11 (NOV), P813-823
ISSN: 0300-5860 Publication date: 20011100
Publisher: DR DIETRICH STEINKOPFF VERLAG, PLATZ DER DEUTSCHEN EINHEIT 25, D-64293 DARMSTADT, GERMANY
Language: German Document Type: REVIEW
Abstract: Dynamic muscular exercise performed by healthy subjects leads to a rise in the left ventricular blood ejection with an acute increase in the local wall shear stress on the endothelium of the arterial vessels. These hemodynamic changes results in a release of endothelium-dependent relaxing factors, one of them concerns nitric oxide (NO). Therefore an arterial vasodilatation with an acute increase in the blood flow volume to the exercising muscle groups occurs. If more than 1/6 of the skeletal musculature is involved in exercise and if training duration exceeds 3-5 hours a week the chronically increased blood flow volume in the cardiovascular system triggers structural and functional changes of the heart and the arterial vessels. It develops a functional intact excentric hypertrophy of the myocardium; within the arterial vessels an increase in the diameter of the muscular arteries supplying the trained muscle groups occurs. These training-induced adaptations of the cardiovascular system are adjusted to improve the aerobic skeletal muscle metabolism.

In congestive heart failure a pathological excentric myocardial hypertrophy is found. In this case the systolic myocardial function is impaired and the left ventricular ejection fraction is reduced already in early stages, so that the cardiac output can not be sufficiently increased during exercise. In addition a dysfunction of the endothelium of the arterial vessels occurs. As a consequence the endothelium-dependent arterial vasodilatation is reduced, so that the peripheral arteries could not supply the muscle groups involved in

exercise with enough blood flow volume. Therefore, the acute delivery of the working musculature with oxygen and energy substrates is insufficient, so that premature muscular fatigue occurs. The reduced exercise resistance of the patients leads chronically to a generalized skeletal muscle atrophy. Ultrastructural analysis revealed a decrease of oxidative type 1 muscle fibers with a relative increase of more glycolytic type 2 fibers. In addition, the volume density and the surface area of the cristae of mitochondria are reduced. All these changes results in a decrease of aerobic skeletal muscle metabolism independent of the blood flow volume, so that the physical fitness of the patients progressively decline. On the basis of the training-induced physiological adaptations of the cardiovascular system, a special exercise therapy supervised by a physician was developed for patients with congestive heart failure NYHA II/III. It have been shown that various exercise programs, which are adjusted to the degree of cardiac function impairment are suitable to restore the endothelial dysfunction of the arterial vessels as well as to cure the disturbed skeletal muscle metabolism in these patients independent of an improvement of cardiac function. Therefore in patients with congestive heart failure NYHA II/III who underwent regularly such an exercise therapy, the secondary impaired physical fitness could be rebuild without an excessive risk for an acute exercise-induced cardiovascular emergency.

...Abstract: cardiovascular system triggers structural and functional changes of the heart and the arterial vessels. It develops a functional intact excentric hypertrophy of the myocardium; within the arterial vessels an increase in the diameter of the muscular...
...Identifiers--TUMOR-NECROSIS-FACTOR; CONVERTING ENZYME-INHIBITION; IMPROVES EXERCISE CAPACITY; NUCLEAR MAGNETIC-RESONANCE; NITRIC-OXIDE SYNTHASE; CARDIAC TRANSPLANTATION; MUSCLE METABOLISM; AEROBIC CAPACITY; CONTROLLED TRIAL; BLOOD-FLOW

10/3,K,AB/19 (Item 3 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

09847322 Genuine Article#: 455JX Number of References: 50
Title: Cyclosporine reduces left ventricular mass with chronic aortic banding in mice, which could be due to apoptosis and fibrosis (ABSTRACT AVAILABLE)
Author(s): Yang GP; Meguro T; Hong C; Asai K; Takagi G; Karoor VL; Sadoshima J; Vatner DE; Bishop SP; Vatner SF (REPRINT)
Corporate Source: Univ Med & Dent New Jersey,New Jersey Med Sch, Inst Cardiovasc Res,185 S Orange Ave,MSB-1576/Newark//NJ/07103 (REPRINT); Univ Med & Dent New Jersey,New Jersey Med Sch, Inst Cardiovasc Res,Newark//NJ/07103; Hackensack Univ,Med Ctr,Hackensack//NJ/07601; Univ Alabama,Birmingham//AL/35294
Journal: JOURNAL OF MOLECULAR AND CELLULAR CARDIOLOGY, 2001, V33, N8 (AUG) , P1505-1514
ISSN: 0022-2828 Publication date: 20010800
Publisher: ACADEMIC PRESS LTD, 24-28 OVAL RD, LONDON NW1 7DX, ENGLAND
Language: English Document Type: ARTICLE
Abstract: A tacit assumption in studies of left ventricular (LV) hypertrophy is that left ventricular/body weight (LV/BW) reflects the extent of myocyte hypertrophy. The goal of the current investigation was to determine if there was another explanation for the reduced LV/BW observed after inhibiting calcineurin with cyclosporine during the development of pressure overload LV hypertrophy as compared with animals that did not receive cyclosporine. Accordingly, we examined the prevalence of fibrosis and apoptosis and measured cell size in the hearts from mice at 1 and 3 weeks after transverse aortic banding with and without chronic cyclosporine. Although LV/BW, compared

to aortic banded vehicle treated mice, was reduced by 30% in aortic banded cyclosporine treated mice, myocyte cross sectional area was similar in both banded groups (346 +/- 9 mum(2) v 336 +/- 13 mum(2)). The volume percent interstitial fibrosis was greater in aortic banded cyclosporine treated animals (1.4 +/- 0.2%) compared with aortic banded vehicle treated animals (0.9 +/- 0.2%, P < 0.05) or in sham animals (0.6 +/- 0.1%), Surprisingly lesions including myocytes containing iron were observed and were most prominent in aortic banded cyclosporine treated animals. Apoptosis, quantitated with TUNEL staining as percent of myocytes, was increased in aortic banded cyclosporine treated animals at 7 days (1.6 +/- 0.4%) compared with aortic banded vehicle treated animals (0.4 +/- 0.1%, P < 0.01) and was still increased at 21 days. Immunoblotting demonstrated a decrease in the phosphorylation of Akt and Bad, and also Bcl-2 levels were reduced in aortic banded cyclosporine treated animals at 7 days compared with aortic banded vehicle treated animals. These proteins protect against apoptosis, and support the concept that cyclosporine inhibited the calcineurin pathway, resulting in enhanced apoptosis, Thus. the decrease in LV/BW in the aortic banded cyclosporine treated animals actually may be due, at least in part, to cell loss and death, as reflected by the enhanced fibrosis and apoptosis and the focal iron deposits in myocyte., (C) 2001 Academic Press.

...Abstract: another explanation for the reduced LV/BW observed after inhibiting calcineurin with cyclosporine during the development of pressure overload LV hypertrophy as compared with animals that did not receive cyclosporine. Accordingly, we examined the prevalence of...
...Identifiers--PRESSURE-OVERLOAD HYPERTROPHY; CARDIAC-HYPERTROPHY; CALCINEURIN ACTIVATION; TRANSPLANTED HEART; CELL-DEATH; RAT HEARTS; METABOLISM; MECHANISMS; EXPRESSION; SURVIVAL

10/3,K,AB/20 (Item 4 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

07568132 Genuine Article#: 182WV Number of References: 32
Title: Pressure overload induces severe hypertrophy in mice treated with cyclosporine, an inhibitor of calcineurin (ABSTRACT AVAILABLE)
Author(s): Ding B; Price RL; Borg TK; Weinberg EO; Halloran PF; Lorell BH (REPRINT)
Corporate Source: BETH ISRAEL DEACONESS MED CTR,DIV CARDIOVASC, DEPT MED, EAST CAMPUS, 330 BROOKLINE AVE/BOSTON//MA/02215 (REPRINT); BETH ISRAEL DEACONESS MED CTR,DIV CARDIOVASC, DEPT MED/BOSTON//MA/02215; HARVARD UNIV,SCH MED/BOSTON//MA/; UNIV S CAROLINA,DEPT DEV BIOL/COLUMBIA//SC/29208; UNIV ALBERTA,DEPT MED, DIV NEPHROL & IMMUNOL/EDMONTON/AB T6G 2M7/CANADA/
Journal: CIRCULATION RESEARCH, 1999, V84, N6 (APR 2), P729-734
ISSN: 0009-7330 Publication date: 19990402
Publisher: LIPPINCOTT WILLIAMS & WILKINS, 227 EAST WASHINGTON SQ, PHILADELPHIA, PA 19106
Language: English Document Type: ARTICLE
Abstract: Cardiac hypertrophy is the fundamental adaptation of the adult heart to mechanical load. Recent work has shown that inhibition of calcineurin activity with cyclosporine suppresses the development of hypertrophy in calcineurin transgenic mice and in in vitro systems of neonatal rat cardiocytes stimulated with peptide growth factors, To test the hypothesis that the calcineurin signaling pathway is critical fur load-induced hypertrophy in vivo, we examined the effects of cyclosporine treatment on left ventricular hypertrophy induced by experimental ascending aortic stenosis for 4 weeks in mice. Left ventricular systolic pressure was elevated to a similar level in aortic stenosis mice that were treated with cyclosporine versus no

drug. Left ventricular mass and myocyte size were similar in treated and untreated aortic stenosis animals and significantly greater than control animals, showing that cyclosporine treatment does not suppress hypertrophic growth. Both treated and untreated animals showed increased left ventricular expression of the load-sensitive gene atrial natriuretic factor, Calcineurin activity was measured in the left ventricle and the spleen from control mice and aortic stenosis mice treated with cyclosporine versus no drug. Levels of calcineurin activity were similar in the spleens of control and untreated aortic stenosis mice, However, calcineurin activity was severely depressed in left ventricular tissue of untreated aortic stenosis mice compared with control mice and was further reduced by cyclosporine treatment. Thus, pathological hypertrophy and cardiac-restricted gene expression induced by pressure overload in vivo are not suppressed by treatment with cyclosporine and do not appear to depend on the elevation of left ventricular calcineurin activity.
...Abstract: mechanical load. Recent work has shown that inhibition of calcineurin activity with cyclosporine suppresses the development of hypertrophy in calcineurin transgenic mice and in in vitro systems of neonatal rat cardiocytes stimulated with...
...Identifiers--ASCENDING AORTIC-STENOSIS; CARDIAC GENE-EXPRESSION; TRANSPLANTED HEART; RATS; MYOCYTES; GROWTH; TRANSITION; RESPONSES; FAILURE; BETA

10/3,K,AB/21 (Item 5 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

07034669 Genuine Article#: 117CW Number of References: 47
Title: High blood pressure management: potential benefits of I-1 agents (ABSTRACT AVAILABLE)
Author(s): Esler M (REPRINT)
Corporate Source: BAKER MED RES INST,POB 6492/MELBOURNE/VIC 8008/AUSTRALIA/ (REPRINT)
Journal: JOURNAL OF HYPERTENSION, 1998, V16, 3 (AUG), PS19-S24
ISSN: 0263-6352 Publication date: 19980800
Publisher: LIPPINCOTT-RAVEN PUBL, 227 EAST WASHINGTON SQ, PHILADELPHIA, PA 19106
Language: English Document Type: ARTICLE
Abstract: Sympathetic nervous system and hypertension Biochemical, electrophysiological, pharmacological and haemodynamic findings support the existence of sympathetic nervous system activation in primary human hypertension. Analysis of regional sympathetic nervous system function, using both neurophysiological methods for measuring sympathetic nerve firing rates, and neurochemical techniques for quantifying regional noradrenaline spillover to plasma has demonstrated activation of the sympathetic nervous outflows to the heart, the kidneys, and skeletal muscle vasculature, particularly in younger patients. The initiating cause of this sympathetic nervous stimulation is unknown, but estimation of central nervous system noradrenaline turnover in hypertensive patients, using measurements of the washout of noradrenaline and its lipophilic metabolites into the internal jugular veins, indicates that activation of forebrain presser noradrenergic nuclei is the probable underlying mechanism.

Consequences of increased sympathetic activity The sympathetic activation present in human hypertension no doubt contributes to the blood pressure elevation, and is a legitimate target for therapeutic intervention with imidazoline receptor-binding agents such as rilmenidine. In addition, the sympathetic nervous activation seems to have adverse consequences in hypertensive patients beyond initiating the blood pressure elevation. There is evidence that neural vasoconstriction has metabolic effects, in skeletal muscle impairing

glucose delivery to muscle, causing insulin resistance and hyperinsulinaemia, and in liver retarding postprandial clearing of lipids, contributing to hyperlipidaemia. Cardiac sympathetic activation is demonstrably a cause of sudden death in heart failure patients; a comparable arrhythmogenic effect is probable in hypertension. A trophic effect of sympathetic activation on cardiovascular growth is also likely, contributing to the development of left ventricular hypertrophy. Rilmenidine, through its central nervous system actions. has been demonstrated to powerfully reduce sympathetic nervous activity in essential hypertension patients.

Inhibiting the sympathetic system As the clinical consequences of sympathetic nervous activation in essential hypertension appear to go beyond that of hypertension pathogenesis, extending to a causal influence in atherosclerosis development, cardiovascular hypertrophy and cardiac arrhythmias, it is possible that, of all antihypertensive drugs, those inhibiting the sympathetic nervous system might best reduce cardiovascular risk. This remains to be tested. I Hypertens 16 (suppl 3):S19-S24 (C) 1998 Lippincott Williams & Wilkins.

...Abstract: A trophic effect of sympathetic activation on cardiovascular growth is also likely, contributing to the development of left ventricular hypertrophy. Rilmenidine, through its central nervous system actions. has been demonstrated to powerfully reduce sympathetic nervous...

...appear to go beyond that of hypertension pathogenesis, extending to a causal influence in atherosclerosis development, cardiovascular hypertrophy and cardiac arrhythmias, it is possible that, of all antihypertensive drugs, those inhibiting the sympathetic...
...Identifiers--SYMPATHETIC-NERVE ACTIVITY; IMIDAZOLINE-PREFERRING RECEPTORS; ESSENTIAL-HYPERTENSION; HEART-FAILURE; GLUCOSE-INTOLERANCE; RILMENIDINE; MILD; TRANSPLANTATION; HYPERTROPHY; MOXONIDINE

10/3,K,AB/22 (Item 6 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

05907306 Genuine Article#: XF682 Number of References: 103
Title: Long-term inhibition of the renin-angiotensin System in genetic hypertension: Analysis of the impact on blood pressure and cardiovascular structural changes (ABSTRACT AVAILABLE)
Author(s): Lundie MJ; Friberg P; Kline RL; Adams MA (REPRINT)
Corporate Source: QUEENS UNIV,DEPT PHARMACOL & TOXICOL/KINGSTON/ON K7L 3N6/CANADA/ (REPRINT); QUEENS UNIV,DEPT PHARMACOL & TOXICOL/KINGSTON/ON K7L 3N6/CANADA/; GOTHENBURG UNIV,DEPT PHYSIOL/GOTHENBURG//SWEDEN/; UNIV WESTERN ONTARIO,DEPT PHYSIOL/LONDON/ON/CANADA/
Journal: JOURNAL OF HYPERTENSION, 1997, V15, N4 (APR), P339-348
ISSN: 0263-6352 Publication date: 19970400
Publisher: RAPID SCIENCE PUBLISHERS, 2-6 BOUNDARY ROW, LONDON, ENGLAND SE1 8NH
Language: English Document Type: REVIEW
Abstract: Objective To compare, using data from published studies, the efficacy of chronic inhibition of the renin-angiotensin system in inducing persistent downregulation of hemodynamic and cardiovascular structural changes in an adult rat with established genetic hypertension with the widely accepted known downregulation in young genetically hypertensive rats,

Study selection We report on 36 studies that satisfied our inclusion criteria (angiotensin converting enzyme inhibitor or angiotensin ii receptor antagonist treatment that lowered arterial pressure levels for at least 3 weeks). Of the 24 studies concerning

developing hypertensive rats, a significant number (n = 17) also examined the persistence of any hemodynamic or cardiovascular effects after withdrawal of treatment, Conversely, of 15 studies using adult rats only seven and three reported on post-treatment hemodynamic and cardiovascular structural indices respectively,

Results During treatment the hemodynamic and cardiovascular structural changes produced were qualitatively and quantitatively similar in the young and adult treated rats, Critical assessment of the persistence of these effects after withdrawal of treatment again found qualitatively similar responses, However, the strength of this finding is limited by the paucity of studies concerning adult rats in which equivalent treatment durations and equipressor doses of treatments were compared between these two age groups,

Conclusions Blockade of the renin-angiotensin system appears to have an efficacy in reversing established hypertension and hypertrophy similar to that with which it prevents the development of hypertension and hypertrophy. This partial 'cure' of hypertension after withdrawal of treatment is clearly evident when treatment is initiated during the development of hypertension and appears to be similar even when treatment is initiated in established hypertension.

...Abstract: efficacy in reversing established hypertension and hypertrophy similar to that with which it prevents the development of hypertension and hypertrophy. This partial 'cure' of hypertension after withdrawal of treatment is clearly evident when treatment is...
...Identifiers--CONVERTING ENZYME-INHIBITORS; LEFT-VENTRICULAR HYPERTROPHY; ARGININE METHYL-ESTER; VASCULAR STRUCTURE; RENAL-TRANSPLANTATION; NORMOTENSIVE RATS; CARDIAC-HYPERTROPHY; ACE-INHIBITION; METHODOLOGICAL CONSIDERATIONS; RESISTANCE VESSELS

10/3,K,AB/23 (Item 7 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

05497681 Genuine Article#: WC267 Number of References: 20
Title: LEFT-VENTRICULAR ADAPTATION TO AORTIC REGURGITATION IN CONSCIOUS DOGS (Abstract Available)
Author(s): GAYNOR JW; FENELEY MP; GALL SA; SAVITT MA; SILVESTRY SC; DAVIS JW; RANKIN JS; GLOWER DD
Corporate Source: CHILDRENS HOSP/PHILADELPHIA//PA/19104; DUKE UNIV,MED CTR,DEPT SURG/DURHAM//NC/27710; DUKE UNIV,MED CTR,DEPT MED/DURHAM//NC/27710; DUKE UNIV,MED CTR,DEPT BIOMED ENGN/DURHAM//NC/00000
Journal: JOURNAL OF THORACIC AND CARDIOVASCULAR SURGERY, 1997, V113, N1 (JAN), P149-158
ISSN: 0022-5223
Language: ENGLISH Document Type: ARTICLE
Abstract: Objective: Cardiac failure as a result of valvular heart disease remains a major clinical problem that frequently leads to ventricular dysfunction, myocardial failure, and even death, The development of irreversible myocardial damage may be especially insidious in volume overload as a result of aortic or mitral regurgitation, Methods and results: Left ventricular wall volume, ventricular function, and myocardial performance were assessed in 10 chronically instrumented conscious dogs before and after creation of aortic regurgitation. Left ventricular wall volume was measured by serial echocardiography. Left ventricular function was assessed by total cardiac output, stroke work, the slope of the Frank-Starling relationship, and the slope of the end-systolic pressure-volume relationship. Myocardial performance was assessed by the slope of the myocardial power output versus end-diastolic strain relationship, End-diastolic wall stress and volume

both increased acutely and remained elevated after creation of aortic regurgitation, Peak systolic wall stress increased initially (1 to 3 weeks) from 336 +/- 30 to 369 +/- 55 mm Hg but returned to control values as left ventricular wall volume increased from 78 +/- 13 to 88 +/- 16 ml after development of compensatory hypertrophy, Left ventricular systolic function remained constant or increased and was maintained initially by increased myocardial performance, which returned to baseline levels after the development of compensatory hypertrophy, Conclusions: Myocardial performance and ventricular function vary independently in aortic regurgitation. Measures of myocardial performance such as the myocardial power output versus end-diastolic strain relationship may be useful in clinical assessment of aortic regurgitation.

...Abstract: values as left ventricular wall volume increased from 78 +/- 13 to 88 +/- 16 ml after development of compensatory hypertrophy, Left ventricular systolic function remained constant or increased and was maintained initially by increased myocardial performance, which returned to baseline levels after the development of compensatory hypertrophy, Conclusions: Myocardial performance and ventricular function vary independently in aortic regurgitation. Measures of myocardial performance...
Research Fronts: 95-4810 001 (LEFT-VENTRICULAR FUNCTION; BRAIN-DEAD ORGAN DONORS; CARDIAC TRANSPLANTATION; CONSCIOUS DOGS; MYOCARDIAL PERFORMANCE; DIASTOLIC DYSFUNCTION)

10/3,K,AB/24 (Item 8 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

03498960 Genuine Article#: PH371 Number of References: 35
Title: RACIAL-DIFFERENCES IN MYOCARDIAL-ISCHEMIA AND CORONARY FLOW RESERVE IN HYPERTENSION (Abstract Available)
Author(s): HOUGHTON JL; PRISANT LM; CARR AA; FLOWERS NC; FRANK MJ
Corporate Source: ALBANY MED COLL,DEPT MED,DIV CARDIOL,A-44/ALBANY//NY/12208; MED COLL GEORGIA,DEPT MED/AUGUSTA//GA/30912
Journal: JOURNAL OF THE AMERICAN COLLEGE OF CARDIOLOGY, 1994, V23, N5 (APR) , P1123-1129
ISSN: 0735-1097
Language: ENGLISH Document Type: ARTICLE
Abstract: Objectives Using invasive measurements of endothelium independent coronary flow reserve and stress thallium testing with or without dipyridamole, this study investigated racial differences in ischemia and coronary reserve in hypertensive left ventricular hypertrophy.

Background. African Americans compared with Caucasian Americans appear to have a higher case fatality from coronary heart disease but lesser amounts of atherosclerotic coronary artery disease. This paradox may be explainable by intrinsic or acquired racial differences in coronary arteriolar autoregulation acid vasoreactivity.

Methods. The study enrolled 98 African and 81 Caucasian Americans referred for cardiac catheterization because of suspected myocardial ischemia but found to have no significant coronary stenosis. Patients H ere stratified by degree of left ventricular hypertrophy for comparison purposes after calculation of indexed left ventricular mass by means of echocardiographic M-mode measurements. Coronary how reserve measurements were made using the intracoronary Doppler catheter and hyperemic doses of intravenous dipyridamole in 100 patients and intracoronary papaverine and adenosine in 72 patients. Seventy-seven percent of patients underwent adequate stress thallium testing with or without dipyridamole.

Results. In African Americans, mean (+/-SD) coronary how reserve decreased from 4.4 +/- 2.3 for 38 without mass hypertrophy to 3.2 +/- 1.3 for 53 with hypertrophy (p = 0.005) to 2.7 +/- 1.1 for 12 with severe hypertrophy (p = 0.02). Thallium testing was abnormal in 31% of those without mass hypertrophy and 59% of those with hypertrophy. In Caucasian Americans, coronary how reserve decreased from 1.1 +/- 2 for 58 without hypertrophy to 3.6 +/- 1.5 for 23 with hypertrophy (p = NS) to 3 +/- 1.5 for 6 with severe hypertrophy (p = NS). Thallium testing was abnormal in 36% without mass hypertrophy and in 39% with hypertrophy.

Conclusions. This study establishes that development of left ventricular hypertrophy in hypertension carries greater physiologic morbidity for African compared with Caucasian Americans, typified by marked reduction in endothelium-independent coronary flow reserve and increased frequency of abnormal thallium tests.

...Abstract: p = NS). Thallium testing was abnormal in 36% without mass hypertrophy and in 39% with hypertrophy.

Conclusions. This study establishes that development of left ventricular hypertrophy in hypertension carries greater physiologic morbidity for African compared with Caucasian Americans, typified by marked...
...Research Fronts: WHITE COAT HYPERTENSION)
92-7073 002 (CORONARY FLOW RESERVE; DOPPLER GUIDED RETROGRADE CATHETERIZATION SYSTEM; ORTHOTOPIC CARDIAC TRANSPLANT RECIPIENTS)
92-0520 001 (LONG-TERM CARDIAC PROGNOSIS FOLLOWING NONCARDIAC SURGERY; CORONARY-ARTERY DISEASE; PERIOPERATIVE...

10/3,K,AB/25 (Item 1 from file: 434)
DIALOG(R)File 434:SciSearch(R) Cited Ref Sci

09799650 Genuine Article#: AY269 Number of References: 45
Title: CARDIAC AND PERIPHERAL VASCULAR-RESPONSES TO ADRENOCEPTOR STIMULATION AND BLOCKADE AFTER CARDIAC TRANSPLANTATION
Author(s): BOROW KM; NEUMANN A; ARENSMAN FW; YACOUB MH
Corporate Source: UNIV CHICAGO,MED CTR,DEPT MED,DIV CARDIOL,CARDIAC NONINVAS PHYSIOL LAB,5841 S MARYLAND AVE/CHICAGO//IL/60637; HAREFIELD HOSP,DIV CARDIOVASC SURG/HAREFIELD/MIDDX/ENGLAND/
Journal: JOURNAL OF THE AMERICAN COLLEGE OF CARDIOLOGY, 1989, V14, N5, P 1229-1238
Language: ENGLISH Document Type: ARTICLE

Title: CARDIAC AND PERIPHERAL VASCULAR-RESPONSES TO ADRENOCEPTOR STIMULATION AND BLOCKADE AFTER CARDIAC TRANSPLANTATION
...Research Fronts: HYPERTROPHY; DIRECTED M-MODE ECHOCARDIOGRAPHY; DIASTOLIC FUNCTION IN MILD HYPERTENSION)
87-4255 001 (LEFT-VENTRICULAR HYPERTROPHY; CHRONIC AORTIC REGURGITATION; BLOOD-PRESSURE DEVELOPMENT IN NEONATAL SPONTANEOUSLY HYPERTENSIVE RATS)

10/3,K,AB/26 (Item 2 from file: 434)
DIALOG(R)File 434:SciSearch(R) Cited Ref Sci

09638331 Genuine Article#: AJ723 Number of References: 50
Title: LONG-TERM FUNCTION OF HUMAN CARDIAC ALLOGRAFTS ASSESSED BY TWO-DIMENSIONAL ECHOCARDIOGRAPHY
Author(s): ANTUNES ML; SPOTNITZ HM; CLARK MB; STEINHARDT MJ; MARBOE CC; SMITH CR; ROSE EA; REEMTSMA K

Corporate Source: CARDIOVASC SURG RES LAB,P&S 17-442,630 W 168TH ST/NEW YORK//NY/10032; COLUMBIA UNIV COLL PHYS & SURG,DEPT SURG/NEW YORK//NY/10032; COLUMBIA UNIV COLL PHYS & SURG,DEPT PATHOL/NEW YORK//NY/10032; PRESBYTERIAN HOSP/NEW YORK//NY/10032
Journal: JOURNAL OF THORACIC AND CARDIOVASCULAR SURGERY, 1989, V98, N2, P 275-284
Language: ENGLISH Document Type: ARTICLE

Research Fronts: 87-2286 003 (CYCLOSPORINE NEPHROTOXICITY; RENAL-TRANSPLANT RECIPIENTS; THERAPEUTIC USE OF AZATHIOPRINE)
87-2588 002 (CARDIAC TRANSPLANTATION; MARKER OF REJECTION; IN-111 ANTIMYOSIN (FAB) IMAGING)
87-4255 002 (LEFT-VENTRICULAR HYPERTROPHY; CHRONIC AORTIC REGURGITATION; BLOOD-PRESSURE DEVELOPMENT IN NEONATAL SPONTANEOUSLY HYPERTENSIVE RATS)
87-6302 001 (END-SYSTOLIC PRESSURE-VOLUME RELATIONSHIP; LEFT-VENTRICULAR...
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882402 21849979 PMID: 11861428

Fabrication of pulsatile cardiac tissue grafts using a novel 3-dimensional cell sheet manipulation technique and temperature-responsive cell culture surfaces.

Shimizu Tatsuya; Yamato Masayuki; Isoi Yuki; Akutsu Takumitsu; Setomaru Takeshi; Abe Kazuhiko; Kikuchi Akihiko; Umezu Mitsuo; Okano Teruo

Institute of Advanced Biomedical Engineering and Science, Tokyo Women's Medical University, Japan.

Circulation research (United States) Feb 22 2002, 90 (3) pe40, ISSN 1524-4571 Journal Code: 0047103

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

6/25

Recent progress in cell transplantation therapy to repair impaired hearts has encouraged further attempts to bioengineer 3-dimensional (3-D) heart tissue from cultured cardiomyocytes. Cardiac tissue engineering is currently pursued utilizing conventional technology to fabricate 3-D biodegradable scaffolds as a temporary extracellular matrix. By contrast, new methods are now described to fabricate pulsatile cardiac grafts using new technology that layers cell sheets 3-dimensionally. We apply novel cell culture surfaces grafted with temperature-responsive polymer, poly(N-isopropylacrylamide) (PIPAAm), from which confluent cells detach as a cell sheet simply by reducing temperature without any enzymatic treatments. Neonatal rat cardiomyocyte sheets detached from PIPAAm-grafted surfaces were overlaid to construct cardiac grafts. Layered cell sheets began to pulse simultaneously and morphological communication via connexin43 was established between the sheets. When 4 sheets were layered, engineered constructs were macroscopically observed to pulse spontaneously. In vivo, layered cardiomyocyte sheets were transplanted into subcutaneous tissues of nude rats. Three weeks after transplantation, surface electrograms originating from transplanted grafts were detected and spontaneous beating was macroscopically observed. Histological studies showed characteristic structures of heart tissue and multiple neovascularization within contractile tissues. Constructs transplanted into 3-week-old rats exhibited more cardiomyocyte **hypertrophy** and less connective tissue than those placed into 8-week-old rats. Long-term survival of pulsatile cardiac grafts was confirmed up to 12 weeks. These results demonstrate that electrically communicative pulsatile 3-D cardiac constructs were achieved both in vitro and in vivo by layering cardiomyocyte sheets. Cardiac tissue engineering based on this technology may prove useful for heart model fabrication and cardiovascular tissue repair. The full text of this article is available at http://www.circresaha.org.

... multiple neovascularization within contractile tissues. Constructs transplanted into 3-week-old rats exhibited more cardiomyocyte **hypertrophy** and less connective tissue than those placed into 8-week-old rats. Long-term survival...

Descriptors: **Heart** Ventricle--cytology--CY; ***Heart** Ventricle --**transplantation**--TR; *Myocardium--cytology--CY; *Temperature; *Tissue Culture--methods--MT

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? s cardio?
      S1 1210002  CARDIO?
? s transplant? or allograft?
          669280  TRANSPLANT?
           92389  ALLOGRAFT?
      S2  697745  TRANSPLANT? OR ALLOGRAFT?
? s s1 and s2
         1210002  S1
          697745  S2
      S3   42476  S1 AND S2
? s develop? (5n)hypertrophy
         4068562  DEVELOP?
          108544  HYPERTROPHY
      S4    5782  DEVELOP? (5N)HYPERTROPHY
? s s3 and s4
           42476  S3
            5782  S4
      S5      39  S3 AND S4
? rd
>>>Duplicate detection is not supported for File 340.

>>>Records from unsupported files will be retained in the RD set.
...completed examining records
      S6      25  RD (unique items)
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